

MC

L3 ANSWER 13 OF 24 MEDLINE
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DOCUMENT NUMBER: 92194468 PubMed ID: 1532214
TITLE: Overexpression of the E1B 55-kilodalton (482R) protein of
human **adenovirus** type 12 appears to permit
efficient transformation of primary baby rat kidney cells
in the absence of the E1B 19-kilodalton protein.
AUTHOR: Zhang S; Mak S; Branton P E
CORPORATE SOURCE: Department of Biology, McMaster University, Hamilton,
Ontario, Canada.
SOURCE: JOURNAL OF VIROLOGY, (1992 Apr) 66 (4) 2302-9.
Journal code: KCV; 0113724. ISSN: 0022-538X.
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LANGUAGE: English
FILE SEGMENT: Priority Journals
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AB To analyze the structure and function of the E1B 19,000-molecular-weight
protein (19K protein) (163R) of human **adenovirus** type
12, mutants were produced at various positions across the 163R-coding
sequence. Viruses bearing mutations within the first 100 or so amino acids
yielded unstable 163R-related products, induced DNA degradation and
enhanced cytopathic effect (cyt/deg phenotype) in KB cells, and
transformed primary rodent cells at much lower efficiencies than wild-type
(wt) virus. Deletion of the final 16 residues at the carboxy
terminus had no phenotypic effect. Alteration of residue 105 reduced
transforming efficiency significantly, suggesting that this region of 163R
is functionally important. Disruption of the AUG initiation codon at
nucleotide 1542 blocked production of 163R completely but resulted in
higher levels of E1B 55K-482R protein synthesis and a transforming
efficiency similar to that of wt virus. These data suggested that while
163R is of some importance, normal transforming efficiencies can be
obtained in its absence if 482R is overexpressed.

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TITLE: p53-independent apoptotic and necrotic cell deaths induced by **adenovirus** infection: suppression by E1B 19K and Bcl-2 proteins.

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CORPORATE SOURCE: Institute for Molecular Virology, St. Louis University Medical Center, Missouri 63110, USA.

CONTRACT NUMBER: CA-31719 (NCI)
CA-33616 (NCI)

SOURCE: CELL GROWTH AND DIFFERENTIATION, (1995 Feb) 6 (2) 131-7.
Journal code: AYH; 9100024. ISSN: 1044-9523.

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AB **Adenovirus** E1B 19K protein prevents premature death of **adenovirus**-infected cells. Viral mutants (19K mutants) defective in the 19K protein induce enhanced cell death, resulting in fragmentation of viral and cellular DNA. The 19K protein can also suppress the effects of certain external cell death-inducing stimuli, such as tumor necrosis factor alpha and various DNA-damaging agents that induce apoptosis. We have examined viral infection of permissive human cells and nonpermissive rat cells to determine if the 19K mutant induces apoptotic or necrotic type of cell death. Infection of normal rat kidney cells with an **adenovirus** type 2 19K deletion mutant induces internucleosomal DNA fragmentation and condensation of nuclear chromatin. Electron microscopic examination of these cells revealed the presence of condensed subnuclear bodies characteristic of apoptosis. In contrast, infection of human A549 cells induces random DNA fragmentation, and these cells do not exhibit characteristic condensation of the nuclear chromatin but contain enlarged nuclei loaded with virus particles. Therefore, it appears that **adenovirus** infection induces both apoptotic and necrotic types of cell death, depending on the cell type. Both types of cell death can be suppressed by E1B 19K protein. Similarly, a recombinant **adenovirus** expressing the human Bcl-2 protein but lacking the E1B proteins can efficiently suppress both apoptotic and necrotic cell death induced by **adenovirus** infection. The requirement of p53 tumor suppressor protein in **adenovirus**-induced cell death was investigated by infection of human Saos2 and mouse p53 nullizygous (p53-/-) cells lacking p53 tumor suppressor protein. (ABSTRACT TRUNCATED AT 250 WORDS)